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A CONTRIBUTION TO THE PATHOLOGY OF SCIATICA.*

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Although sciatica is one of the commoner forms of nervous disease, it is rarely that an opportunity has been afforded to study the underlying pathologic changes, more especially as the affection itself is never a menace to life. In the literature of this subject, which has now attained enormous dimensions, one is surprised to find on what meager anatomic facts our knowledge of the pathology is based.

I have been able to collect only 11 cases in which the condition of the nerve was noted postmortem, the majority of these observations emanating from the older writers.

Of this number, three only received histologic study. During the past 15 years, a period distinguished by great activity in the field of neuropathology, I can find no record of a postmortem examination.

The knowledge gained from the study of these few cases, has been further supplemented by the inspection and palpation of the nerve trunk during the operation of nerve stretching, formerly so popular, and the occasional examination of excised fragments of the sheath.

Clinically speaking, sciatica may occur in two forms: The neurotic, a pure neuralgic form, in which pain is practically the sole symptom, and the neuritic, in which other symptoms are present, indicating inflammation of the sheath and involvement of the nerve fibers.

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That the nerve in this group of cases is actually the seat of structural alterations, may be inferred from the tenderness and the accompanying motor, sensory, and vasomotor disturbances, usually slight in degree, but often persisting for a considerable time or even enduring permanently.

To this latter group must be reckoned the great majority of cases, so that the socalled sciatica is regarded as an interstitial neuritis or perineuritis.

Sciatica, as is well known, has its analog in the upper extremity in the brachial neuralgia or the brachial perineuritis, and Hutchinson has even described an optic neuritis of gouty origin.

The relationship existing between these forms of neuritis and the gouty and rheumatic diatheses, has long been recognized clinically, and was first emphasized by English observers.

It is supposed that the fibrous sheath of the nerve trunk suffers in the same manner as do the fibrous structures of the muscles and joints, constituting a neuritis of gouty origin.

One English writer, Buzzard,[1] has even gone so far as to attribute the symptoms to a local deposit of sodium urate in the perineural lymph spaces. This attractive theory, however, has never been confirmed by actual observation.

It is interesting in this connection to recall the finding of a fine powdery deposit of sodium urate in the smaller nerves surrounding old gouty joints by Charcot and others. These nerves lay in the midst of tissues saturated with sodium urate and the deposits were evidently secondary.

Before proceeding to a detailed description of my own case, I will summarize briefly the more important facts gleaned by postmortem study of the nerve by other observers.

The first description is found in the classic monograph of Cotugno,[2] published in 1764, who noted an edematous infiltration of the nerve sheath, from which 15 cc. (½ oz.) of serum was removed by puncture. Chaussier,[3] in 1803, found the nerve enlarged and more voluminous than normal, the perineurium edematous, and the bloodvessels of the sheath dilated and varicose.

In 1818, Martinet[4] described the nerve as edematous and of a reddish hue, with hemorrhages into the sheath. In another case the nerve was enlarged (hypertrophied) and infiltrated with a serosanguineous fluid.

Gendrin[5] (1826), in a case of relapsing sciatica, found the affected nerve more voluminous and of a reddish hue as compared with that of the sound side; the redness and swelling extending from the sciatic notch to the popliteal space. In the upper portion, the nerve fibers were separated by a sanguinolent edema which could be expressed by pressure, rendering the nerve substance less dense. The cut end of the nerve trunk was also of a reddish hue.

In another case of 15 days' duration, Gendrin found the nerve trunk from the plexus to the popliteal space, reddened, swollen, and the seat of a serous infiltration. Its volume was increased threefold in comparison with the sound side. On section the white nerve bundles stood out on a reddish background.

Rousset[6] (1819), in a sciatica of 14 years' duration, found the nerve sheath looser in texture than normal, and the veins of the perineurium dilated, but without evidences of edema or sclerosis.

In a case recorded by Gubler and Robin[7] (1860), of old sciatica with atrophy, the nerve was carefully examined macroscopically and microscopically with entirely negative results.

Hasse[8] (1864) simply states that he examined the nerve in a case of sciatica without finding any material change.

Leudet[9] (1873), in a case of old sciatica without atrophy, describes the nerve as more voluminous than on the sound side, with thickening of the sheath and an increase of fat in the interfibrillary spaces. The nerve fibers were unaltered.

Fernet[10] (1878), records a typical case of 15 days' duration. The nerve was of a reddish hue, and the vessels of the neurilemma were injected. The volume of the nerve trunk was increased, giving it a cylindric appearance, so that when laid upon a table it did not flatten out, as that of the sound side. The consistency was also increased, and firmer to the feel, so that on palpation the individual nerve bundles were not felt so distinctly as on the sound side. These changes were most apparent at the point of exit from the sciatic foramen.

The microscopic examination was carried out in the Laboratory of Vulpian, by Déjérine and Raymond. Both nerves were examined without detecting any evident alterations in the nerve sheath or its fiber. Fernet maintained, however, even in the absence of histologic changes, that the gross appearance of the nerve clearly indicated a neuritis.

To recapitulate briefly: From the year 1764 to 1878, 11 pathologic observations have been recorded. Of this number, 3 were negative, including 1 case with microscopic examination (Gubler et Robin). In 8 cases the result was positive, the nerve was found enlarged, reddened, and edematous. This group included only two microscopic examinations, both negative, that of Leudet, who simply described the nerve fibers as unaltered, and that of Fernet (Vulpian's laboratory), in which careful examination failed to reveal any histologic changes.

CASE REPORT—SUMMARY.

A man aged 59, presenting the typic symptoms of sciatica of one month's duration, affecting the left leg. Death due to croupous pneumonia. The nerve trunk in the popliteal space and below the sciatic notch was distinctly enlarged and thickened. This increase in size due to the deposit in the epineurium and epineural fat of a firm translucent substance of gelatinous consistency. Histologic examination of the nerve trunk was entirely negative.

History.—The patient was a laborer by occupation. He had indulged freely in alcohol. In November, 1902, he had an attack of sciatica on the left side, ushered in by lumbago, all symptoms disappearing in about 6 weeks.

In the latter part of February, 1903, sciatic pains recurred in the left leg, typical in character and distribution, and very severe. He applied for treatment at the neurologic clinic of the Cornell Medical School.

At this time movements of the leg were attended by great pain and the sciatic phenomenon was exquisitely marked.

The usual tender points were present along the course of the nerve, a spot of especial acuteness marking the exit of the sciatic from the pelvis. The achilles jerk was diminished on the left side. The knee-jerks were present and equal.

There were no demonstrable objective disturbances of sensibility, although a feeling of numbness along the outer side of the left leg was remarked by the patient.

In the posterior muscles of the left thigh, buttock and left leg, irregular muscle waves and coarse fibrillary twitchings were noted, the socalled myokymia. The urine was free from albumin and sugar. Signs of general arteriosclerosis were apparent, otherwise the examination was negative.

As the symptons were severe and acute, he was referred to Bellevue Hospital, Cornell Medical Division, for treatment and was admitted to the service of Dr. W. Gilman Thompson. After 3 weeks' hospital treatment, the symptoms abating in their intensity, the patient was allowed to return home.

During the next two weeks he visited our clinic twice and except that the intensity of the pain had subsided, presented practically the same symptoms as were noted in the first examination.

Both on standing and walking the left leg was favored. It was held slightly flexed at the knee in the erect posture, with the left gluteofemoral furrow directed upward and to the right. No curvature of the spine was noted.

Active myokymic twitchings of the whole posterior aspect of the left leg and buttock were visible, increased by exposure and irritation of the skin and by percussion of the muscles; also by the recumbent posture.

Similar twitchings, although of lesser degree, were present in the homologous muscle group of the opposite side.

The left achilles jerk was considerably diminished in intensity and the tendo-achillis in the recumbent posture appeared relaxed on the affected side (Oppenheim's symptom). Tender points were still present along the course of the nerve.

The left thigh and leg had undergone a slight but visible atrophy. Although paresthesia along the outer side of the left leg still persisted, no objective disturbances of sensibility were demonstrable.

A few days later the patient developed croupous pneumonia and was readmitted to Bellevue Hospital in the service of Dr. Wm. K. Draper, dying March 23, 1903.

A complete autopsy was refused but permission was granted to examine the affected nerve.

Pathologic Examination.—The left sciatic and internal popliteal nerves were exposed throughout the greater part of their course.

From the sacrosciatic notch downward for a distance of about 8 inches, the trunk of the nerve is thicker and more voluminous than normal. This enlargement in the circumference of the nerve is due to the deposit in the perineural areolar tissue and fat of a substance which in feel and appearance resembles a firm gelatin. A similar deposit is noted in the popliteal space above the bifurcation, extending upward for several inches. The bifurcation itself, the internal popliteal, and that portion of the sciatic occupying the middle of the thigh present a normal appearance.

In those portions of the nerve trunk occupied by the gelatinoid deposit, signs of inflammation, such as redness or congestion, are wanting.

This increase in the circumference of the nerve is not due to an edema, which drips or may be stripped out after removal, but is dependent upon a firm, homogeneous, translucent substance deposited in the epineurium and having the consistency of a firm jelly. No urate crystals could be demonstrated in the fresh state.

Microscopic sections of various levels of the sciatic and internal popliteal nerves from the sciatic notch to the internal malleolus were prepared according to the following methods: Osmic acid, counterstained with Rubin S., hematoxylin and eosin, Van Gieson, Marchi, Weigert, Weigert-Pal and counterstain, and the Weigert fibrin stain. In none of the sections examined were there any evidences of round-cell infiltration or other signs of acute inflammation. Those portions of the nerve, the seat of the gelatinous swellings, presented no evident histologic changes, the epineurium containing only areolar tissue and fat of normal appearance.

No fibrin could be demonstrated in these areas by the Weigert fibrin stain.

The nerve fibers, so far as could be determined, were free from any degenerations.

The perineural connective tissue of the individual nerve bundles is thickened and coarse.

The arteries of the sheath, both the larger vessels of the epineurium and perineurium, and the smaller vessels of the endoneurium, are markedly sclerosed and thickened, but nowhere occluded.

Small extravasations of blood are found in the loose areolar tissue of the sheath at all levels examined. These do not stand in immediate relation to the bloodvessels, and were probably artificially produced in removal.

Portions of the biceps, gastrocnemius, flexor longus digitorum adjacent to the nerve trunk were removed and subjected to histologic examination, with negative results.

Remarks.—In the foregoing case there can be no question as to the diagnosis of sciatica. The onset with lumbago, the relapse, and the subsequent course and symptoms are typical.

The case was also of an intensity and duration sufficient to warrant the full development of inflammatory or other structural changes if such exist in this affection. We find only a firm, jelly-like deposit in the nerve sheath, quite structureless histologically, and unaccompanied by inflammatory changes.

The sclerosis of the neural bloodvessels and the thickening of the nerve sheath are natural concomitants of the age of the patient and the general arteriosclerosis.

It will be remembered that in 8 of the 11 recorded cases the nerve was noted as enlarged and thickened ; 3 of these subjected to histologic examination revealed no structural alterations.

Furthermore, the three cases described as negative were of many years' duration, when any transudate or active inflammatory changes would naturally have subsided; they also date from a period (1819, 1860, 1864) when pathologic technic was far from perfect. I think we must therefore recognize a perineural deposit as the essential and underlying condition in this group of cases, in which the ordinary cellular evidences of inflammation are entirely wanting.

The nature of this transudate and its origin are still uncertain, but a large mass of clinical evidence indicates a probable relationship to the gouty and rheumatic states.

As is well known, the fibrous sheath of the nerve contains lymph-spaces and channels lined by endothelium and continuous with the subdural and subarachnoid cavities, thus furnishing the histologic elements of a serous membrane, so that one might speak of a *neural gout* or *neural rheumatism.*

The further elucidation of this problem belongs properly to the field of chemic pathology.

A pathologic anatomy such as has been described harmonizes very well with the clinical facts; acute course and eventual complete restoration of function, the symptoms resulting from compression of the nerve fibers and the nerves of the sheath (nervi nervorum).

The acceptation of a lesion of this nature, does not however exclude the possibility of a true interstitial inflammation, with cellular infiltration, organization and permanent damage to the nerve.

It would seem fitting, however, to discriminate between this form, which is properly speaking a neuritis, and that under discussion in which the sheath is the seat of a structureless deposit of obscure origin.

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